# A REVIEW OF ACUTE INTESTINAL OBSTRUCTION

BY

P. J. W. MONKS

*The Royal Infirmary, Bristol*

Acute intestinal obstruction is a common and serious surgical emergency. Vick (1932) reported a mortality of 26 per cent in a collected series of nearly 7,000 cases, including 322 cases from Bristol Royal Infirmary. This high mortality has been reduced in this series to 16 per cent, but this still seems a high figure although it is in keeping with a recently reported series by Smith, Perry and Yonehiro (1953), and is also the figure quoted in *British Surgical Practice* (1948). This high mortality could undoubtedly be reduced by earlier diagnosis.

Between January 1952 and June 1957, 5,131 emergency operations were carried out in the general surgical department of the Bristol Royal Infirmary; 440 of these operations were for acute intestinal obstruction and these cases are now reviewed. Table 1 shows the main causes of obstruction and sets out the number of cases in each group and their respective mortality. For comparison, Vick's figures of 1932 are shown where these are comparable.

TABLE I

Acute intestinal obstruction: showing the main causes in 440 cases, and the mortality in each group. The mortality is compared with the larger series published by Vick, 25 years ago.

| Cause of Obstruction | Cases | Deaths | % Mortality | % Mortality Vick 1932 |
|---|---|---|---|---|
| Femoral hernia | 104 | 6 | 6 | 15·9 |
| Inguinal hernia | 99 | 10 | 10 | 11·5 |
| Ventral hernia | 27 | 7 | 25 | 38 |
| Total external hernia | 230 | 23 | 10 | — |
| Obstruction with no mention of strangulation | 65 | 13 | 20 | 32·9 |
| Obstruction with strangulation | 67 | 2 | 3 | 34 |
| Colon obstruction | 45 | 21 | 47 | — |
| Post-operative | 25 | 6 | 25 | — |
| Vascular | 8 | 6 | 70 | — |
| Total | 440 | 71 | 15·5 | 26·2 |

## EXTERNAL HERNIA

Strangulated external hernia was the commonest cause of obstruction and occurred in 230 cases, just over half of the total, and there were 23 deaths (10 per cent). The site of the hernia had a very direct bearing on the mortality, and important differences in each group of herniae were apparent.

Strangulated femoral hernia occurred 104 times, and resulted in 6 deaths (6 per cent), strangulated inguinal hernia 99 times causing 10 deaths (10 per cent). Femoral

hernia presented at a later age than inguinal hernia (see Fig. 1), the youngest case was 45 and the highest incidence was in the 70–79 age group; inguinal herniae were found at all ages and the peak incidence was in the 60–69 age group. Age seemed

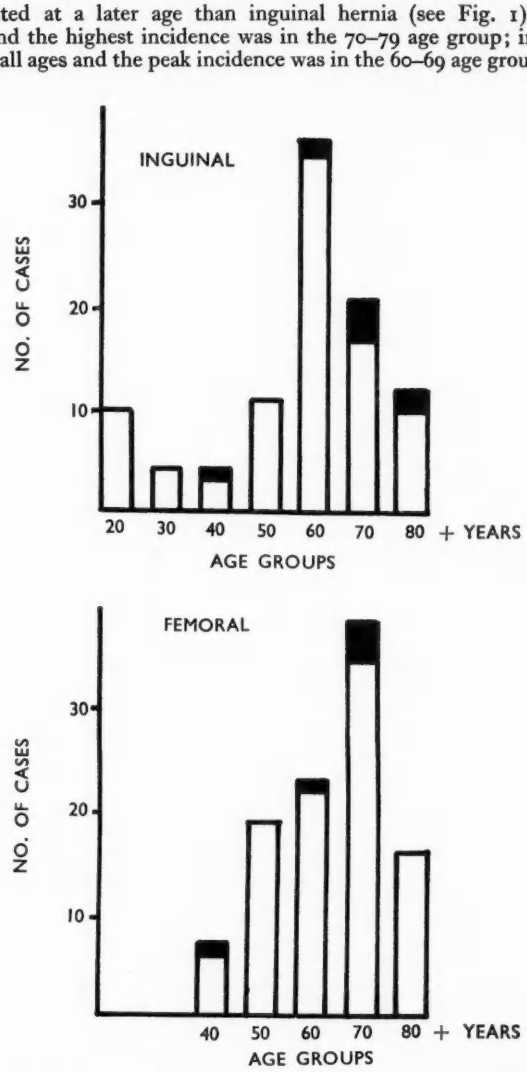



FIG. 1. *Compares the age incidence in cases of strangulated inguinal and femoral herniae. The black areas represent deaths.*

to have little effect *per se* on the mortality of strangulated femoral hernia for none of 16 cases over the age of 80 died, and only 4 of the 55 cases over the age of 70 years. In the inguinal hernia group, however, age was an important factor for there were 7 deaths in 33 cases over the age of 70.

All the deaths from strangulated femoral hernia occurred in late cases, all were desperately ill on admission and in spite of vigorous intravenous fluid replacement all

the deaths occurred within 24 hours of operation, and all were due to peripheral circulatory failure. By comparison the deaths from strangulated inguinal hernia occurred in both early and late cases (see Fig. 2), and the cause of death was different:

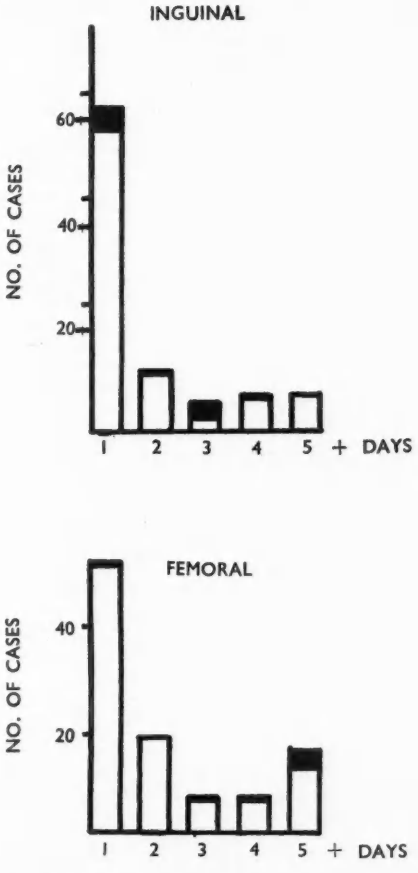



FIG. 2. *Compares the length of history in cases of strangulated inguinal and femoral herniae. The black areas represent deaths.*

only 1 patient died of peripheral circulatory failure shortly after operation, (this was the youngest death, a man of 45 who had been vomiting severely for 5 days, and was in gross electrolyte imbalance on admission); the other deaths were all in the elderly and were mainly due to associated cardio-respiratory complications, and occurred later in the post-operative period: one patient died of heart failure, 1 of a cerebro-vascular accident and 3 of pulmonary embolus; the other deaths were more directly due to the obstruction, for 1 inhaled vomit whilst still in the casualty department, 1 old man of 90 had total gangrene of the whole small bowel, and 1 died of a leaking suture line following a resection.

Resection was required in 25 cases of strangulated femoral hernia. Twelve of these had been strangulated for over 3 days, one for 26 days, and 3 of these died; the others with a shorter history all survived. Three cases requiring a resection had a history of less than 12 hours; one of these was a Maydl's hernia with the strangulated loop lying within the abdomen. Only 9 cases of inguinal hernia needed a resection and 2 died. There were 2 cases of reduction-en-masse of an inguinal hernia, the patient in both cases reducing the hernia himself.

Femoral hernia seems to carry a lower mortality than strangulated inguinal hernia in spite of the older average age, higher resection rate, and longer average history. The reason for this would seem to lie in the difficulty of reducing and repairing an often large inguinal hernia in an elderly patient with associated cardio-respiratory disease. These patients had often been refused elective surgery for their hernia because of their poor general condition. In comparison the femoral hernia is usually small and it is relatively easy to relieve the strangulation. About half these cases were approached from below the inguinal ligament, thus no muscles were cut and an easy convalescence ensured.

Strangulated ventral and umbilical hernia had a very high mortality, for there were 7 deaths in 27 cases (25 per cent). Five cases required resection and three of these died. This very high mortality rate was in part due to the attempted repair of very large herniae, for 2 cases died within 24 hours of operation with gross respiratory insufficiency, and 1 case died of a burst abdomen on the seventh post-operative day. These patients were often very obese and several were diabetic; these factors were important contributory causes of death.

### OBSTRUCTION WITHOUT MENTION OF STRANGULATION

There were 65 cases of small bowel obstruction without strangulation, and 13 cases died (20 per cent). The commonest cause of obstruction in this group was adhesions; there were 40 such cases. The adhesions were all multiple and often very extensive, most followed previous operations. Other causes were metastases, 7 cases; appendicitis (either retro-ileal or with a pelvic abscess), 6 cases; gall stone ileus, 4 cases; simple stricture, 2 cases; carcinoma, 3 cases; simple tumour, Crohn's disease, Meckel's diverticulum, 1 case each. Two of the deaths were due to multiple metastases. Appendicitis, gall-stone ileus and carcinoma of the jejunum caused 1 death each. The remaining 8 deaths were all due to multiple adhesions.

These cases of so-called "simple" obstruction frequently had an insidious onset, often with previous minor attacks of colic and vomiting. Many had several days history by the time they reached hospital (see Fig. 3). Only 9 cases had a history of less than 24 hours and a third had a history longer than 5 days. As a result many were ill and dehydrated—sometimes severely so—by the time they reached hospital, and treatment for several hours with intravenous fluids was necessary before they were considered fit for surgery.

The diagnosis of established intestinal obstruction was fairly obvious by the time these patients reached hospital, although the cause of the obstruction was not always clear. Many of these cases had plain supine and erect X-rays of the abdomen taken on admission, these showed various degrees of intestinal dilatation associated with fluid levels and confirmed the diagnosis of obstruction in nearly every case. However they were of little value in demonstrating the cause of obstruction or the presence of strangulation.

Difficult operations to relieve these obstructions were sometimes necessary, especially in those cases with extensive adhesions. Two cases had such dense adhesions that it was considered only possible to do an enterostomy between dilated and collapsed bowel. Three cases developed further obstruction post-operatively and required a second operation. The lateness of these cases, together with the sometimes formidable operations in the presence of gross intestinal dilatation, account for the high mortality.

A few of these cases were treated by gastric suction and intravenous fluids for several days; this led to further delay and the impression is that these cases did not do so well as those operated on as soon as their fluid balance had been restored.

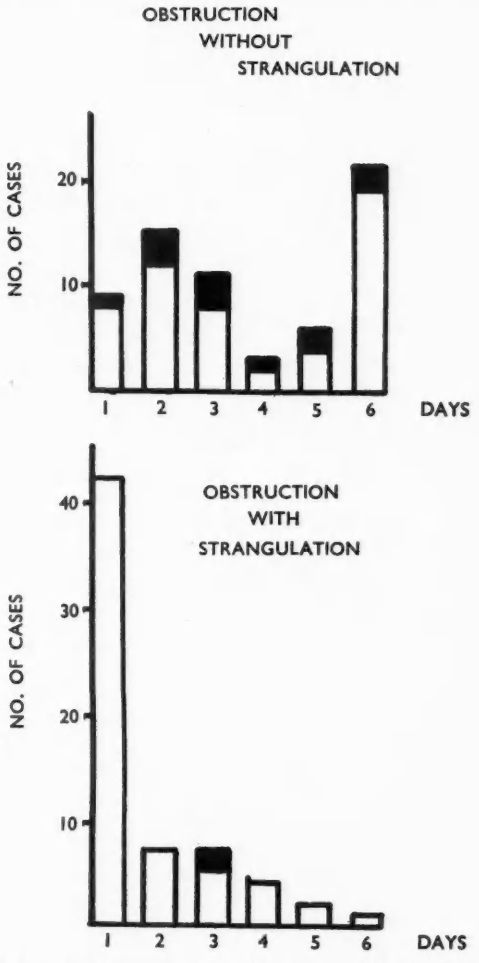



FIG. 3. *Compares the length of history in cases of obstruction with and without strangulation. The black areas represent deaths.*

OBSTRUCTION WITH STRANGULATION

This group had the lowest mortality of all, there were 65 cases and 7 required resection, and there were only 2 deaths (3 per cent). One patient died of congestive cardiac failure on the seventh post-operative day, and the other case was misdiagnosed as a dissecting aneurysm and the strangulation was only found post-mortem. The commonest cause of strangulation was a band, often single, this was found 46 times.

Other causes were—internal hernia 9 cases, intussusception 9 cases, volvulus of the small gut 2 cases, Meckel's diverticulum 1 case.

The initial severe pain brought these patients to hospital much earlier than those without strangulation. Fig. 3 illustrates this point; the great majority of cases were admitted within 24 hours and many within a few hours of the onset of the pain. In nine cases there was considerable delay in diagnosis after the patient was seen in hospital, the most common provisional diagnosis was renal colic. One case was so diagnosed for five days before laparotony showed the true state of affairs, and another thought to be a dissecting aneurysm until post-mortem examination showed a loop of small bowel strangulated by a simple band. The severe pain is commonly referred to the back, probably due to involvement of the mesentery, and it is all too easy to interpret this as renal pain. Cases with a tight strangulation may only vomit once in the early stages and often the bowel acts after the onset of the pain. The early physical signs are minimal, there is no distension, little rigidity, and sometimes very little tenderness. This lack of physical signs in the abdomen together with a negative X-ray examination lead to the difficulty in diagnosis when these cases are first seen.

Plain X-rays of the abdomen were taken in 30 of these cases. No evidence of obstruction was reported in 6, paralytic ileus in 2, subacute or partial obstruction in 9, and complete obstruction was only reported in 13 cases or just under half. The negative findings were all in early cases, for in these the small bowel has not had time to distend and the colon and rectum still frequently contain gas. Whilst fluid levels and dilated loops are characteristic of a small gut obstruction that has been present for 24 hours, a relative absence of gas is often the most important sign of an early strangulation. Plate XVIII shows a plain film of the abdomen 2 hours after the onset of pain, there is an almost complete absence of gas in the small bowel, gas is present in the colon and there is a suggestion of a soft tissue mass. At laparotomy several feet of small bowel were found tightly snared by a band, the bowel above the obstruction being quite empty.

Major small bowel strangulation rapidly produces shock, from loss of blood and plasma into the bowel lumen, the bowel wall, and the peritoneum. In the later cases this must be replaced as rapidly as possible prior to operation. Occasionally cases are seen when the shock develops very early, is often profound within an hour or two, and responds poorly to adequate blood and plasma transfusions, until the strangulation is relieved. In these cases it is best to operate almost as soon as an efficient plasma drip is running for dramatic improvement was particularly well seen in the two extremely ill cases of complete small bowel volvulus, where the blood pressure returned to normal as soon as the volvulus was untwisted.

### POST-OPERATIVE OBSTRUCTION

There were 25 cases of intestinal obstruction developing after operation, whilst the patient was still in hospital. This is a serious complication for 6 (25 per cent) of these patients died. These cases are divided into 2 groups, the first arising within 48 hours of the primary operation, the second after a period of well being, usually about the seventh post-operative day.

Some of these cases were due to internal hernia or volvulus, and they had severe abdominal pain. Others were the result of organizing adhesions and followed a period of post-operative ileus, one condition gradually merging into the other. It was in these latter cases that most difficulty was experienced in deciding the right time to re-operate.

In the early post-operative group there were 8 cases, two of these were not recognized and died 48 hours and 9 days post-operatively. In both cases at post-mortem gangrenous bowel due to a small bowel volvulus was found. The other cases were all treated by gastric suction and intravenous fluids for a variable time, usually for several days (1 case for as long as 14 days) before it was decided to re-operate.

In the later group there were 17 cases and 4 of these died. Four cases developed sudden severe colic and vomiting; this led to almost immediate laparotomy and all did very well. The remaining cases developed more insidiously and were treated at

## PLATE XVIII

*A supine X-ray of the abdomen of a young man with a two hour history of internal strangulation. There is an almost total absence of gas, yet at operation 4 feet of strangulated small bowel was found.*

first by g
operation
the other
post-oper
by gastric
the avera
areas rep
increase v



FIG. 4. *Sh*
*on t*
*The figur*

The m
one. It w
primary s
tion only
once any

Acute
cent). T

first by gastric suction. This treatment was continued for periods up to 14 days; operation then became imperative. Four of these died, one was not operated on and the others died within 24 hours of the second operation. Fig. 4 shows all the cases of post-operative obstruction arranged in columns according to the length of treatment by gastric suction before the second operation, the height of each column representing the average length of stay in the hospital after the second operation and the shaded areas representing the deaths. It is clearly shown that the morbidity and mortality increase with delay in relieving the obstruction.

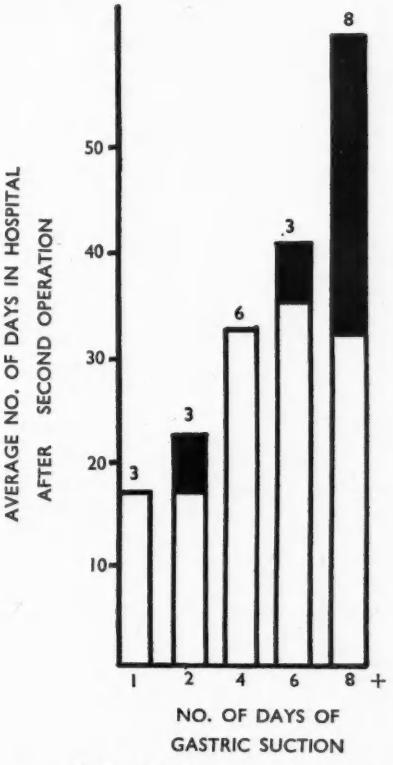



FIG. 4. *Shows how the average stay in hospital after the second operation increased with the time spent on treatment with gastric suction before operation in cases of poor operative obstruction. The figures at the end of each column represent the number of cases. The black areas denote deaths.*

The management of these patients is difficult, and the decision to re-operate a serious one. It would appear that, especially when obstruction develops several days after the primary surgery, early re-operation would be a wise course. Continued gastric aspiration only seems to disguise an established obstruction and the patient will deteriorate once any initial dehydration has been corrected.

### LARGE BOWEL OBSTRUCTION

Acute large bowel obstruction occurred in 45 cases and there were 21 deaths (47 per cent). This high mortality may be spurious, because some other cases may have been

relieved by simple measures and then have had an elective operation with a better prognosis. Carcinoma of the colon was the commonest cause. There were 32 such cases, 19 of these being situated in the sigmoid, 10 in the transverse, and 3 in the right colon. There were 6 cases of volvulus, 2 of diverticulitis, and 1 each of carcinoma of the bladder, simple stricture and stricture of a colostomy. In addition there were 2 cases who both died with gross abdominal distension and faecal impaction following the prolonged use of morphia.

Many of these patients were elderly and had other serious concomitant disease; several of the elderly refused treatment when that meant a colostomy. Many of the carcinoma cases were very ill on admission with anaemia and dehydration from long-standing obstruction. Fifteen (50 per cent) of the obstructions due to carcinoma died, one death followed a later resection but all the others died as a direct result of their obstruction, complicated often by associated cardio-respiratory disease, or metastases.

A few of the early cases of neoplastic obstruction were treated by primary resection but most by laparotomy and the establishnemt of a colostomy. It would seem that even this is too large a procedure in the very poor risk case and a blind caecostomy might be used more often. This could be followed by a more satisfactory colostomy or a resection as soon as the patient is fit.

Sigmoin volvulus in 1 case required resection and this patient survived. Two other cases died, one as the result of a further volvulus and the other who was very ill on admission was treated by rectal decompression but died shortly afterwards. The other 4 cases were all treated by laparotomy, untwisting the volvulus and the passage of a rectal tube.

### VASCULAR CAUSES OF OBSTRUCTION

This is an uncommon but important cause of obstruction because of the unsatisfactory methods of treatment and the poor prognosis. There were only 8 cases but 6 of these died. According to the Registrar General's Report (1955) this is an increasing cause of death, the crude death rate per million having increased fourfold between 1945 and 1955. Three patients had mesenteric emboli and three mesenteric arterial thrombosis; these cases were all explored and infarction of all the bowel supplied by the superior mesenteric artery was found. None of these cases had the vessel explored, and they all died shortly after the operation.

In the early stages after mesenteric embolus there are very few signs to be found in the abdomen and the patient remains surprisingly well for several hours. The diagnosis is therefore difficult, but should be considered in any patient with auricular fibrillation who has an attack of severe abdominal pain. Early diagnosis should lead to exploration of the superior mesenteric artery for successful embolectomies have now been reported (Shaw and Rutledge, 1957).

The 2 cases that survived were probably cases of mesenteric venous thrombosis, as both had a previous history of venous thrombosis in other veins. These 2 cases were treated by intravenous heparin and antibiotics, and if the non-operative diagnosis of venous thrombosis could be made with confidence then this is probably the best method of treatment.

### CONCLUSIONS

A consecutive series of 440 cases of acute small and large bowel obstruction has been presented. Seventy-one (15·5 per cent) cases died, some of severe associated disease, but many as a direct result of the obstruction. The factor still chiefly responsible for the high mortality is delay in diagnosis.

The mortality rate has improved from 26 per cent given by Vick for the period 1925–1930, but the improvement is not in proportion throughout the series. The prognosis has improved most in the cases of internal strangulation, for the mortality rate has improved from 34 per cent in 1932 to 4 per cent in this series. Strangulated femoral hernia has also shown a marked improvement from 16 per cent to 6 per cent,

but obstruction from non-strangulating adhesions has only changed from 32 per cent to 20 per cent. The mortality of strangulated inguinal hernia and vascular obstruction remains the same.

The improvement in the cases of internal strangulation is in part at least due to earlier admission to hospital. Most of Vick's cases were admitted on the second day but in this series two thirds of the cases reached hospital on the first day. The average length of history in the cases without strangulation has not changed to the same extent and there has consequently been less improvement. Similarly there has been an increase in the proportion of femoral herniae reaching hospital on the first day, but the length of history of the inguinal herniae remains about the same. The prognosis has improved in the former but remains constant in the latter.

Since 1930 great advances in fluid and electrolyte therapy, blood and plasma transfusion, antibiotics and anaesthesia have taken place. These have all contributed greatly to the improved prognosis, but their value lies in the management of the late case admitted after several days of vomiting, the case who is dehydrated, distended, and in urgent need of resection. These aids are rarely needed in the early case.

The most important cause of the high mortality in intestinal obstruction remains delay in operating. The delay is brought about firstly by difficulty in diagnosis, but it is also sometimes prolonged by the use of the gastric suction tube for periods longer than are required for rehydration.

Vick in 1932 said, "If we accept the contention that by early diagnosis and treatment these people may be saved from miserable death then these mortality rates are too high." This statement remains true today, for many of the deaths in this series could have been prevented by an earlier diagnosis.

## REFERENCES

*British Surgical Practice*, 1948. London, Butterworth and Co.
Registrar General's Report, 1955.
Shaw, R. S. and Rutledge, R. H. 1957. *N. England J. Med.*, **257**, 595.
Smith, G. A., Perry, J. F., and Yonehiro, E. G., 1955. *Surg. Gynae. Obst.*, **100**, 651.
Vick, R. M. 1932. *Brit. Med. J.*, **2**, 546.

# SQUINT

BY

CALBERT I. PHILLIPS,

*Ophthalmologist, United Bristol Hospitals.*

The investigation and treatment of strabismus take up a high proportion of the ophthalmologist's time. Its treatment is rewarding for the patients are often children in whom the prevention of amblyopia, restoration of binocular vision and improvement in appearance will be of great benefit for the remainder of their lives. The assessment of a diplopic patient is an interesting exercise and may contribute much to a neurological diagnosis.

The patient, if adult, usually complains of blurred or double vision. A great deal of time and trouble are often saved if uniocular diplopia be excluded from the start. If his blurred vision disappears when he closes each eye separately, then his diplopia is binocular. Many patients with astigmatism or early cataract complain of "double vision" (this, in a sense, is true, though "blurred vision" would be more correct) which, of course, remains in the affected eye(s) when the other is closed. The patient can try out this test on the spot so that binocular and uniocular diplopia can be immediately differentiated.

The first aim in diagnosis is to decide whether a patient has an (a) incomitant or (b) concomitant squint. Usually the former are adults complaining of double vision, and the latter children. The term "incomitant" implies that the visual axes of the two eyes are not *both* pointing towards the object of attention *in all directions* of gaze. A paralysis or paresis of, say, the right lateral rectus muscle will result in mis-alignment when the patient looks to the right and therefore diplopia: there is no abnormality in the other directions. The term "concomitant" strabismus means that the visual axes maintain the same, abnormal, relation to each other in all directions of gaze. The visual axis can be regarded as a line drawn from the macula through the centre of the pupil.

## COVER TEST

Before history taking, aetiology and mechanisms are discussed, it is convenient to describe this important part of the examination. The cover test proper is an objective means of determining the presence of squint and is particularly useful for children with concomitant squints. Various refinements are used but the form to be described is the basic one for the detection of a manifest squint—whether it is concomitant or incomitant. See Plate XIX. Tell the patient to look at an object e.g. the tip of a pen nib held at about arm's length from his eyes, and to keep looking at it. Now cover the right eye, but watch the left; if the left does not move, it is not, and was not, squinting. Next uncover the right eye and tell the patient to look at the pen again (both eyes being uncovered). Then cover the left eye but watch the right; if the latter does not move to take up fixation, no manifest squint is present—in either eye. The younger the child the less he will co-operate and often the interpretation of the test is difficult. A child's interest may be maintained if he is told to look at a flashing torch light or a small brightly coloured toy. Each eye should have at least fair visual acuity—a blind eye will not move to take up fixation though it may be squinting.

Often, in spite of a negative cover test, observant parents may insist that the child has a squint. Either the squint is intermittent or some other condition simulates it. The usual cause of "apparent squint" is epicanthic folds at the inner canthi which produce an appearance of convergence of the eyes—see Plate XX.

(*a*)

(*b*)

(*c*)

(*d*)

FIG. 1. *Cover test.* (*a*) *The patient is told to look at a small object held in the examiner's hand about a yard away.* (*b*) *The right eye is covered and the examiner notes that the left eve moves outwards to take up fixation i.e. the left eye has a convergent squint.* (*c*) *Both eyes are uncovered again and object is re-fixed.* (*d*) *The left eye is covered but no movement occurs in the right eye i.e. it does not squint.*

Diagnosis. *Left convergent squint. Since movements of both eyes were full, "concomitance" is proved. The squint was "cured" by the correction of the hypermetropia with glasses.*

PLATE XX

FIG. 2. *Epicanthic folds simulating convergent squint. Cover test negative.*

## I. INCOMITANT STRABISMUS

Careful history-taking is as vital in ophthalmology as in any other branch of medicine.

Its duration and possible intermittency having been ascertained, the next important question is whether diplopia is vertical or horizontal. "Are the two images side by side or one above the other—or both?" "Does the separation increase when you look right, left, up or down; near or far?" From this information, if the patient is observant, a good indication of the affected muscle can be obtained. Horizontal diplopia only to the right suggests a (right) abducent nerve palsy, common in elderly people, which often recovers within a few weeks and is usually attributed to a "vascular" cause—a rather vague explanation which implies either pressure on the nerve by an atheromatous intracranial artery or blockage of a nutrient vessel. These symptoms *could* be due to a left medial rectus palsy but in isolation this is rare, even in disseminated sclerosis.

Ocular movements should be examined. It is easier to assess the range of movement of each eye separately, the one not under observation being covered by the examiner's hand or an envelope etc. Very often a muscle may be only paretic so that no obvious impairment of movement exists. Clinically, the affected muscle may be discovered by the following system. The patient is asked to look at, say, a pen nib with both eyes open and to watch it as it is moved upwards, downwards, to his right and left, and also to the intermediate points. The area in which maximum separation of the doubled images occurs is ascertained. Suppose the separation to be vertical, and maximal downwards and to the (patient's) left. Which eye and which muscle is affected? The image which is further away from the centre (the lower in this position) always belongs to the paretic eye so. . . . To the patient looking at the pen nib down and to his left: "Close your left eye and tell me which image disappears". . . . "The upper". Therefore the *right* is the affected eye since the further-away-from-centre image belongs to it. Now, the main action of the superior oblique muscle is depression of the eye when it is already adducted towards the nose, therefore the right superior oblique is the affected muscle—a common occurrence.

However, often the results are equivocal, and the ingenious "Hess screen" examination is very useful in such cases. Over the patient's eyes are placed goggles, a red glass before the right and a green before the left. He is seated one metre from a black screen over which are scattered, at regular intervals, red dots or red lights. Now, the right eye can see these *red* dots but the left cannot (because the green glass allows only green rays to pass, not red). Into his hand he is given a pointer with a green ring at its tip and he is told to place the green ring (see only by the left eye) round the red dots. If he has, say, a left lateral rectus paresis, the right eye will look at each red dot but, when the gaze is directed towards the patient's left, the left eye will not have moved far enough and therefore the green tip of his pointer will be placed some distance short of the dots in that region. However, when he looks up, down and to the right, his eyes will be "comitant" and he will place the green ring over the red dots successfully. A chart is completed from which can be seen at a glance the direction in which one eye falls short and therefore which muscle is paretic.

Once the weak or paralysed muscle has been detected, the cause remains to be diagnosed. A general history and examination, especially of the other cranial nerves, should not, of course, be omitted. Isolated palsies of extra-ocular muscles are, however, common—for example in dissemated sclerosis, myasthenia gravis (unilateral ptosis is also common and may be quite sudden in onset: variability is important) and diabetus mellitus (peripheral neuropathy which often involves the trigeminal nerve also). In the attempt to locate and diagnose the lesion, it is most useful to trace the nerve, in imagination, from the muscle supplied (hereditary ocular myopathy may, rarely, occur; "thyrotropic" infiltration with lymphocytes and fibrous tissue is usually accompanied by proptosis) to its nucleus and consider what neoplastic, inflammatory, vascular etc. causes may affect it en route.

### *Ocular Torticollis*

A child or an adult may have an abnormal posture of the head for a variety of reasons, including "fibrosis" of a sternomastoid, abnormalities of the vertebrae etc. One rather uncommon cause is a congenital weakness of an extra-ocular muscle, usually a superior oblique or a superior rectus. The cause is unknown, but birth injury is often blamed; some cases have been shown to be due to poor developmental differentiation of the muscle or to abnormal fibrous bands which limit movement of an eye. A child with such a disability, if it affects a muscle which moves the eye up or down, may be able to overcome his vertical diplopia and build up good stereoscopic reflexes by (a) holding his head backwards or forwards to relieve a paretic elevator or depressor respectively, (b) tilting his head over towards the shoulder on the same side as the eye with the higher *image* and (c) turning his head so that the responsibility for elevating and depressing the affected eye is transferred to another set of muscles. Which of these tricks is employed by the patient will depend on the affected muscle. A right lateral rectus palsy will result merely in a turn of the head to the right. The common congenital paresis of a right superior rectus muscle will be compensated by a backward tilt of the head, a turn to the right to transfer the onus for elevating and depressing the eye to the oblique muscles (which act mainly on the adducted eye) and finally, a tilt towards the right shoulder. This last element in the posture has two advantages: (a) by postural and vestibular reflexes it compensates for the abnormal height of the right eye's image and (b) it compensates for the outward wheel-rotation of the *other* eye produced by the synergist of the *right* superior rectus, which is the *left* inferior oblique. In adult life, compensation may sometimes begin to break down and the patient complain of intermittent vertical diplopia. Photographs taken in childhood may show that he has had a head tilt all his life—and may be the only foundation for an accusation of superciliousness (elevator palsy with nose in the air) or suspiciousness (depressor palsy with head bent forwards, eyes looking "under his brows").

### *Treatment*

If the symptoms are severe enough and there is no chance of further spontaneous improvement (e.g. 6–9 months after a head injury) then operation may be considered. Ophthalmologists are not usually enthusiastic in transplanting the outer (or inner) halves of the superior and inferior recti to the insertion of a paralysed lateral rectus muscle though some good results have been reported; a weakening, by recession of its insertion, of the medial rectus on the same side is usually more certain to produce improvement. In such a case, recession of the *other* eye's medial rectus (a synergist of the opposite lateral rectus) will reduce the area of gaze in which diplopia is suffered, for it is over-acting in response to the redoubled efforts of the brain to force the eyes to look to the side of the paralysed muscle. Even in a complicated case of unilateral third cranial nerve palsy improvement can be achieved; if ptosis is effectively eliminating the diplopia, however, operation may be best avoided. Considerable improvement in symptoms due to a paresis of a superior oblique muscle can be obtained by myectomy of the inferior oblique on the same side, along with recession of the over-acting contralateral synergist, the inferior rectus.

## II CONCOMITANT SQUINT

This occurs especially in children and the main factor in the very common "accommodative concomitant convergent squint" is hypermetropia. Another common type is the weakness of convergence (i.e. latent, or intermittently manifest, divergent squint for near objects) which often affects the elderly presbyope or the student hard at his last-minute reading for an examination. In either case, after the history of age of onset, intermittency etc. has been obtained, it is vital to check that there is a full range of movement in each eye separately before concomitance is accepted.

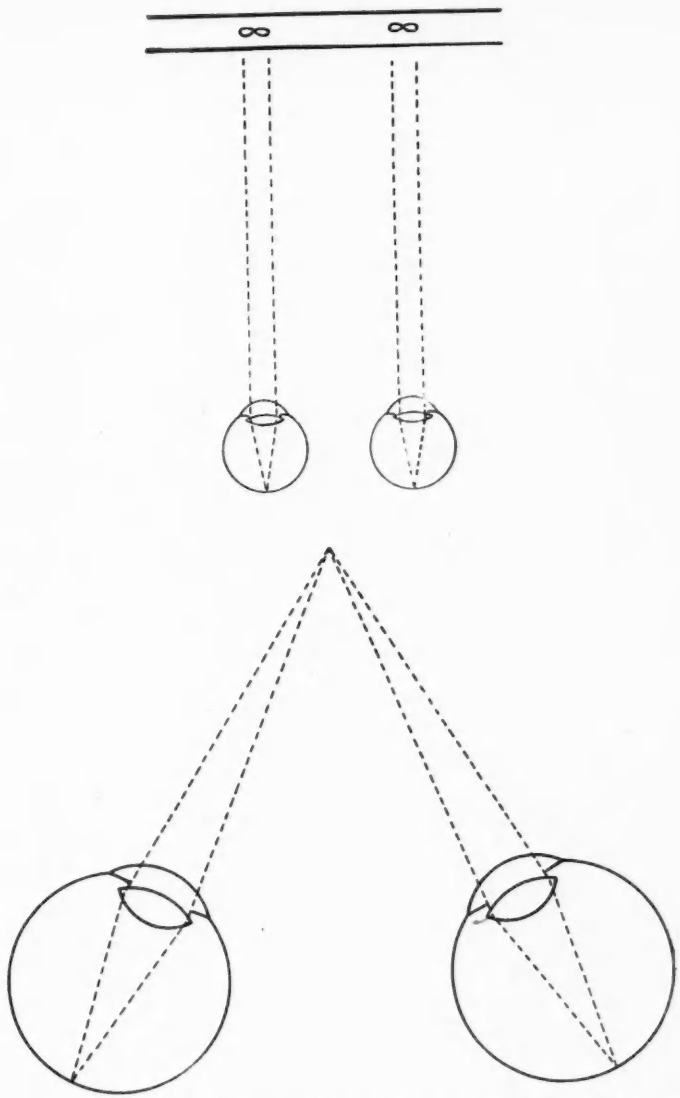



FIG. 1. *Upper. On a small scale, emmetropic eyes look at an infinitely distant object. Visual axes almost parallel.*

*Lower. On a larger scale, emmetropic eyes look at a near object by (a) accommodating to bring divergent rays to a focus on the maculae and (b) converging by a proportionate amount to avoid diplopia.*

A typical symptom in accommodative squint in children is that the squint is worse, or even only present, when the child looks at a near object. The following is a brief account of the mechanism:

Normal eyes are emmetropic, i.e. no accommodation is required for distance. In order that an eye can produce a clear image of a near object, the ciliary muscle must contract (accommodation) which makes the curvature of the surface of the crystalline lens greater. In addition, if diplopia for near objects is to be avoided, each eye must be turned nasal-wards ("converged") enough to make the two visual axes intersect the object. See Fig. 1. Now, the brain has a mechanism for "gearing" accommodation to convergence in *normal* eyes. But if the patient is born with hypermetropic eyes (i.e. his lens system is too "weak" for the length of his eyeball) he must accommodate *even for distant* objects and, therefore, to a greater degree than normal for near. Unfortunately, the gearing mechanism will therefore tend to produce over-convergence i.e. a convergent squint, unless the harmonic reflex can alter the ratio of the gears. Fortunately it often succeeds, because all hypermetropes do not squint.

Many cases quickly develop a constant squint, however. If the two eyes are equally hypermetropic and see equally well, the child may choose to "alternate" i.e. use either eye indiscriminately, allowing the other to converge. But if one is slightly more hypermetropic than the other or has astigmatism or some other abnormality causing blurred vision (e.g. chorioditis due to toxoplasmosis in the macular region: note the implication here that a convergent squint may be a presenting symptom of unilateral disease of the eye) then the child will use the better eye consistently and neglect the other, which becomes permanently convergent. The danger now is that the child, suffering diplopia, suppresses the image from the squinting eye (cf. the practised microscopist who can keep both eyes open, suppressing the eye not in use), ***a condition which in children quickly becomes permanent***, hence amblyopia ex anopsia. There follow then two reasons for *early* investigation of strabismus i.e. as soon as it has appeared. Early treatment of hypermetropia with spectacles (even a child of 18 months may be induced to wear spectacles) may "cure" the squint and prevent the need for operation. The sooner an amblyopic eye is treated (a) with spectacles if necessary, and (b) by occlusion of the other eye, the less chance it has of suffering permanent impairment of vision. If a convergent squint still remains after that treatment then recession of the medical rectus/recti is usually done, sometimes with resection of the lateral recti. Thus, two or more operations at different times may well be required.

The presbyope must almost invariably wear glasses for reading in order to replace the deficiency in accommodation. In addition, many develop weakness of convergence, with a complaint not only of blurring of individual letters on a page but that, intermittently, "the letters run into each other" i.e. horizontal diplopia. Mild degrees respond to convergence exercises which consist in their simplest form, of looking at, say, some small print (wearing the reading correction) and repeatedly approximating it to the eyes. Such an exercise should be done for 5 minutes three times daily: a week usually is sufficient. More severe degrees may require the incorporation of base-in prisms in the reading glasses.

Strabismus, in the widest sense of the term, is common and affects patients of all ages. Treatment is a matter of constant careful supervision, in which operations, if required, are merely incidents.

# TRANSPORTING SEVERELY PARALYSED PATIENTS

BY

JAMES MACRAE, M.D.
*Ham Green Hospital, Bristol*

The transport of patients suffering from diseases which impair breathing or swallowing, or both, is a problem which has become of great importance during the past decade. This is because most of these patients suffer from poliomyelitis, and the recently established respiratory centres now provide a much better service and are able to save the lives of many of these desperately ill patients. The spectacle of a racing ambulance carrying a patient whose unassisted vital capacity is ebbing is now an unnecessary drama. During the years 1947 to 1954 there were nine such wild drives which ended in this hospital with a dead patient: one of these, a young man from Bridgwater appeared to have died in the ambulance a mile away; a boy of sixteen died in his own home six miles from Ham Green before the arrival of an ambulance which was stationed at Weston-super-mare. During these years only one patient was transferred to us from another hospital under conditions of planned transport. She arrived in a tank respirator driven by a precarious system of batteries and transformers. She was accompanied by eight men and two nurses; everything and everybody travelled in a furniture van. After such a valiant effort it was a pity that the patient inspired vomit during the move and died as a result within the day.

With the introduction of artificial positive pressure respiration in 1953 and an increasing experience of the management of cases suffering from respiratory insufficiency, a better and safer method of transport was obviously necessary. Thus, in 1955, we thought it a duty of this hospital to provide a service designed to get these patients safely into our respiratory unit.

Initially the plan seemed to have three elements:

(*a*) The reporting of cases;
(*b*) Sending assistance and equipment to the patient;
(*c*) Transporting the patient to Ham Green.

We imagined that this hospital would provide doctors, nurses and equipment ready to go to the patient at short notice and we thought we might need to do tracheotomy, set up intravenous drips, give nasal-gastric tube feeds and possibly nurse patients at home or in another hospital for a few days before transfer was possible. Equipment in accordance with these ideas was assembled and proved to be very bulky. The provision of a specially equipped ambulance was mooted but the idea was discarded as impracticable. From the beginning, we decided to depend on manual methods of artificial breathing and so have never attempted to carry mechanical respirators for emergency cases.

The availability of this new transport service was advertised to Medical Officers of Health by the Regional Hospital Board and it was specially requested that the doctor in charge of the case should get in direct personal touch with doctors in this hospital whenever the service was required. It was considered that we would need a discussion with the doctor in charge in order to assess the needs of the case and to give advice which he could apply in maintaining the patient until the team from Ham Green could arrive. Undoubtedly this telephone conversation has remained the most important item of the emergency service.

Our first call for assistance came from Exeter on an August Bank holiday afternoon. The information received by telephone suggested a severe case of bulbar poliomyelitis, very nearly dead already. Hence, since our team could hardly get to Exeter in less than three or four hours, it was suggested that a local anaesthetist be called to insert an

endo-tracheal cuffed tube quickly and breathe the patient manually on the journey to Ham Green. This was in fact done very successfully and the patient arrived in remarkably good condition so that we could safely perform tracheotomy and maintain ventilation by a mechanical positive pressure machine. This first experience pointed to a fresh conception of the problem of transporting such patients: supervision and management should be undertaken by a local anaesthetist.

The hospital team is still in being; there are two doctors in this team without nurses; this has proved quite sufficient. We discovered that the amount of equipment can be much reduced. What is required for these patients consists of:

(*a*) Equipment for posture in an ambulance, so we carry a simple, adjustable, inverted V frame for cases of simple swallowing defect;
(*b*) Equipment for manual mask ventilation for cases of breathing insufficiency only;
(*c*) Equipment for passing a cuffed endotracheal tube and manual ventilation thereafter for combined swallowing and breathing deficiency.

A limited assortment of drugs and instruments are carried, especially pentothal and scoline with sterilized syringes. A sucker and oxygen cylinder completes the equipment. In all, these materials weigh about fifty pounds and are easily carried by hand. The sole purpose of the team is now confined to the reasonably quick movement of the patient to hospital, and the providing of adequate artificial respiration on the way.

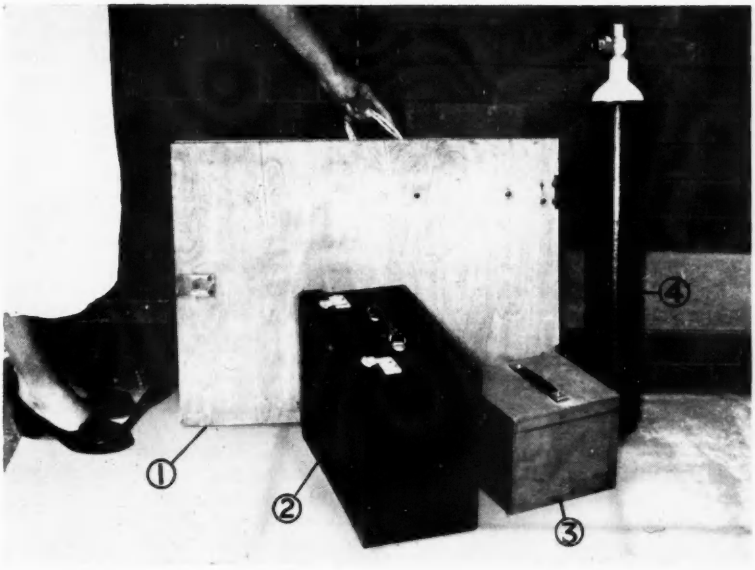
Some twenty-six patients have actually been transported into hospital since 1955. Nineteen have been cases of poliomyelitis; five were cases of infective polyneuritis, one of acute ascending myelitis and one with myasthenia gravis. Apart from one child with pneumonia we have not been sent any unsuitable cases. These patients have come from Exeter, Truro, Bideford, Taunton, Yeovil, Cheltenham, Gloucester, Swindon, Bath and Bristol. In eight cases our own team went out to collect the patient and in all of the others we enlisted the aid of a local anaesthetist. The help that these anaesthetists have given has been invaluable, although as they themselves have commented the job required is what they have to do every day—"breathe" a patient by hand through a mask or through an endotracheal tube. In some cases this assisted ventilation was unnecessary, but the anaesthetist was there in the ambulance to help if required. It has been found that once the patient's airway is clear and manual ventilation established, the patient is safe and can be transported long distances without incident. The journey can be quite unhurried; there is no need for a specially equipped ambulance, and suction is seldom required if the patient's airway is reasonably dry at the beginning of the journey.

One of the advantages of the initial telephone talk with the doctor in charge of the case, has been that a decision can be made whether a local anaesthetist should be called in. Then a short talk with the anaesthetist has always resulted in him understanding completely what is required so that the patient is brought to us competently and without fuss.

The results have been very good. There have been no deaths in an ambulance since the service started and all the patients transported have arrived in hospital in good condition.

We employ the Oxford Inflating Bellows as a method of hand ventilation, using it either through a face mask or through an endotracheal tube. It is a most efficient instrument which only needs the addition of a pressure gauge in order to avoid too great ventilation pressures.

## PLATE XXI




1. *Adjustable, inverted "V", bed-frame.*
2. *Case containing instruments, drugs, etc.*
3. *Oxford inflating bellows.*
4. *Oxygen cylinder.*

APL

*A*

*Prof*
us the
*Dr.*
illness.
in her
she co
gave h
of wha
and sh
She
respira
faint y
No cli
present
oedem
emboli
The
heart n
monia
a high
Lab
fluid,
improv
develo
althou
steadil
How
going
cent. S
this di
weekly
on 18
white
nuclea
Ster
is a m
deficie
crease
It w
some
never
per ce
were 3
On
cells.
chest,
red bl
was ne

## APLASTIC ANAEMIA WITH POST-TRANSFUSIONAL HAEMOSIDEROSIS

*A Clinical Pathological Conference of the University of Bristol Medical School*

CHAIRMAN: PROFESSOR T. F. HEWER

*Professor Hewer:* I will ask Dr. Macrae, who first looked after this patient, to give us the clinical history.

*Dr. J. Macrae:* This lady, aged 63, had no previous hospital admission or serious illness. Two months before admission to Ham Green Hospital she had had thrombosis in her left leg and recovered satisfactorily after bed rest. Three weeks before admission she complained of breathlessness on exertion, her heart beat was irregular, her doctor gave her digitalis and she improved. Some three days before admission she complained of what she called "a cold" with cough. She had pain in the right side of the chest and she coughed up blood stained sputum.

She was admitted on 27th July 1957 and was found to have a temperature of 99·2°F; respirations 30 per minute; pulse 88, highly irregular and fibrillating. There was a faint yellowish tinge to the skin but no definite jaundice. Blood pressure was 115/80. No clinical enlargement of the heart was noted but a small right pleural effusion was present. The sputum was flecked with blood, the right leg was rather discoloured, no oedema was present, and the clinical picture suggested that she had had a pulmonary embolism causing pulmonary infarction on the right side.

The chest was X-rayed and the effusion confirmed with some enlargement of the heart noted in addition, and it seemed possible that there were some patches of pneumonia at the left base. The patient was very constipated and she felt much better after a highly successful enema. She was treated with rest, penicillin and digitalis.

Laboratory investigation showed the haemoglobin to be 77 per cent and the pleural fluid, which was brownish in colour, contained no carcinoma cells. She appeared to improve but on the 2nd August she complained again of severe right-sided pain and developed a pleural rub at the left base and we rather assumed that she had had another, although smaller, pulmonary infarction. Thereafter her pulmonary condition improved steadily and did not enter into the clinical picture again.

However, during the same time it was noted that haemoglobin percentages were going down steadily and, by the middle of September, the haemoglobin was 53 per cent. She had been receiving ferrous sulphate orally since the end of August, but, as this did not seem to improve the haemoglobin, she received "Imferron" 5 c.c. twice weekly from 10th September onwards to a total of eight doses. The blood picture on 18th September showed: haemoglobin 52 per cent; red blood cells 2,500,000; white blood count 5,200; polymorphs 68 per cent; lymphocytes 22 per cent; mononuclears 7 per cent; eosinophils 3 per cent.

Sternal marrow was obtained on 20th September and Dr. Bolton reported: "This is a moderately cellular marrow. It is normoblastic and shows no evidence of iron deficiency or of megaloblastic haemopoiesis. The peripheral blood film shows increased red cell regeneration".

It was suggested that the patient was suffering from concealed haemorrhage of some sort, but we could find no site of haemorrhage; in particular occult blood was never found in the stools, although tested many times. Serum bilirubin was 1·4 mgms. per cent; blood urea 33·3 mgms. per cent, and on 23rd September 1958 reticulocytes were 3·7 per cent and the haemoglobin was 53 per cent.

On 2nd October 1957 the patient was transfused with one and a half pints of packed cells. Shortly after transfusion she developed some small patches of purpura on the chest, buttocks and legs. The haemoglobin on 9th October 1957 was 70 per cent with red blood cells 3,010,000; white blood count 6,000; platelets 30,100. Coombs's test was negative.

The patient felt much better; she had got tired of being in hospital and wanted to go home. Although we had still found no reason for her anaemia, we allowed her to go and I told her general practitioner that she would probably need further investigation and we would have her back.

On 30th October 1957 she was re-admitted, pale, breathless with a haemoglobin of 38 per cent; platelets 41,000 per cu. mm. She was given a transfusion of one pint packed cells and transferred to the care of Dr. Cates at the Bristol Royal Infirmary on 6th November 1957 as a case of probable aplastic anaemia.

*Dr. J. E. Cates:* When she came in we had the full history from Dr. Macrae, we checked on one or two points, and turned to the patient. We found that she was indeed very pale, fat and poorly. She was short of breath even when turning over in bed. Though she was apathetic, she retained her sense of humour. She was covered with purpura over her shoulders and chest. Temperature was between 99 and 100°F. Her pulse and respiration were normal. There was bruising on her arm where blood pressure had been measured. There was no jaundice, a feature you might find in haemolytic anaemia. There were no enlarged lymph nodes, and no enlarged spleen to fit in with a diagnosis of leukaemia. Her heart was slightly enlarged with a raised venous pressure, some peripheral oedema and rales at the bases. Her haemoglobin was 42 per cent, E.S.R. 69 mm/hour. Total white cell count 1,700 (polymorphs 600), and reticulocytes below 0·6; platelets 10,000/cu. mm. and a normochromic film. The stools contained occult blood; we interpreted this as meaning that she had some scattered oozing of blood from her bowels. All this seemed to fit in with Dr. Macrae's suggestion that her bone marrow was failing. White cells and platelets were reduced suggesting that she had panmyelophthisis. There was no bleeding to cause anaemia and no evidence of haemolysis.

A further bone marrow biopsy was done by Dr. Bolton but the report was not conclusive.

We then tried to find out why she should have developed marrow aplasia. We telephoned Dr. Macrae and asked what her treatment had been. We were interested to know if the patient had had chloramphenicol or sulphonamides for her pneumonia or if she had had any chlorpromazine.

An important danger to bear in mind is benzene, for there is always the possibility of washing paint or clothes in it; we found no story of any toxic substance. We wondered if this marrow aplasia could be due to something else, for example secondary to uraemia. She did not look uraemic clinically. There was no acidosis and the laboratory findings excluded this possibility. We wondered about D.L.E. because she had a high sedimentation rate but we found that she had no other clinical evidence to support this diagnosis. Thus her heart was normal, her urine was normal, and there was no arthritis. Plasma proteins were not much abnormal and the L.E. test was negative on several occasions.

We transfused her with blood at the rate of 4 pints in the first three days; we had to watch her very carefully as she was in heart failure and rapid transfusion is likely to kill people if they have heart failure. We also gave her mersalyl and kept her propped up until she had a high haemoglobin.

In the next 4 weeks in hospital she had a total of 8 pints and was obviously very much improved by it. She developed a chest infection at both bases so she was given a course of antibiotics and improved. She then had a tender right calf and pain in the chest and we thought this was a pulmonary embolism. She stopped fibrillating soon after admission, but this began again. We gave her two more pints and she then went home for Christmas; she was admitted again shortly afterwards and in January she had two more pints and in February we stopped transfusing her.

In February before she actually died she became very drowsy and on 17th February had two fits and her respiration rate became raised. It was noticed that she had a right upper motor facial nerve weakness at that time. Next day she had some more fits and spasmodic twitching of the right arm. On the 19th she developed right-sided hemi-

plegia; both toes went up but it was more definite on the right; she then had a series of fits and died in coma. We presumed she had some form of haemorrhage into the brain.

Summing up, this was an old lady of 63 who had a chest infection and developed a progressive anaemia after this with haemorrhages and low platelet count, low reticulocyte count and a hypoplastic marrow. We thought that this aplasia was "ideopathic". Survival was prolonged by repeated blood transfusion and antibiotics. We noticed towards the end that besides being anaemic she was rather earthy in colour, but we did not investigate this to see whether the blood we had given her was doing anything odd.

*Professor Hewer:* You say that there were no drugs given at all which might have had a bearing on the aplasia. In the notes it said that 15 grams of sulphatriad were administered at Ham Green.

*Dr. Cates:* I cannot imagine Dr. Macrae giving her sulphatriad.

*Dr. Macrae:* No, we didn't.

*Dr. M. E. H. Halford:* Was there any evidence that she had destroyed the transfused blood?

*Dr. Cates:* We thought the rate of destruction when transfusion stopped was about as fast as one would expect it normally to be. There did not seem to be any haemolysis. People having blood transfusions do tend to destroy the older cells in the bottle rapidly. Her haemoglobin fell to 35 from 80 per cent in about 5 weeks, about the rate one would expect with somebody not making any of her own. The mean survival of transfused red cells is 60 days, if given fresh.

*Dr. O. C. Lloyd:* Can you give us some idea about how often you meet such a case and how often do you have to admit that it is idiopathic?

*Dr. Cates:* In about half the cases.

*Dr. G. H. Tovey*: Yes, in about 50 per cent.

*Professor Hewer* describing the post mortem: This woman had a great many petechiae and bruises. The cause of death was easy to determine, it had already been envisaged clinically. There was a massive subdural haemorrhage on both sides, slightly more on the left than the right. There was also subarachnoid haemorrhage. The haemorrhage probably came from superficial veins. There was haemorrhage around the base of the brain and it was considerably compressed by haemorrhage. I think this caused the coma. There were no haemorrhages inside the brain on section. There were no vascular lesions. The whole of the neurological findings and the cause of death could be attributed to the haemorrhage.

The bone marrow, as you would have expected, was extremely pale: there was no red marrow in femur, sternum, ribs or vertebrae. Histologically a few cellular areas were found but these consisted only of groups of lymphocytes and plasma cells. This fits in with the haematological findings during life.

The right pleural cavity was entirely obliterated by old fibrous adhesions, with a solid mass of scar tissue just over the diaphragm and another, with some brown patches of haemosiderin, over the lower lobe posteriorly. In this lower lobe was a pulmonary infarct that was becoming absorbed and there were several patches of bronchopneumonia. Near the hilum of the lung was a partially organized thrombus attached to the wall of a pulmonary artery. This was a nice confirmation of Dr. Macrae's diagnosis of pulmonary embolism. There was also an extensive terminal bronchopneumonia.

The liver had some cough grooves on its surface and since Dr. Macrae says she had no cough until she was admitted to hospital it is interesting to observe that these grooves could be produced within seven months. The liver also had a rusty colour as a result of the presence of a great deal of haemosiderin in the portal zones of the liver and in the Kupffer cells. There was no scarring. Iron was also present in the spleen, pancreas, thyroid and pituitary and in many other situations: nowhere had it provoked any fibrosis.

There were many submucous haemorrhages in the stomach but none in the intestines.

I think one can recapitulate the course of events to some extent. She originally had some venous thrombosis in her legs and this caused several pulmonary emboli. They were probably associated with some low grade pulmonary infection. She then developed an aplastic anaemia as a result of total aplasia of the bone marrow. The cause of this marrow aplasia is unknown, but it may have been due to some drug she obtained from a chemist: we have no evidence. The blood transfusions and iron injections accounted for the haemosiderosis. The absence of megakaryocytes in the bone marrow probably accounted for the fall in blood platelets and her numerous haemorrhages helped to dispose of some of the blood that she was receiving. Eventually spontaneous intracranial haemorrhages caused death.

*Dr. Cates:* To go back to sulphatriad, I think we rang up the family doctor and asked how the bad cold had been treated, and I think that is where it came in.

*Dr. Coles:* Novalgin, which contains amidopyrine, is an antipyretic which is, I believe, available at some grocers. Did she have this?

*Professor Hewer:* That is a very nice suggestion of Dr. Coles's—that she took the drug to relieve the pain caused by venous thrombosis and the drug caused her anaemia. Unfortunately we have no evidence that she took any such drug.

*Dr. Cates:* That is so: we obtained no evidence that she had in fact had any amidopyrine.

# Notes and News

New Year Honour: G. C. Kelly, C.B.E. (Civil Division).

Professor J. M. Yoffey has been awarded the order of "Knight of Dannebrog" for his services to Danish medical students.

Professor G. Gordon Lennon has been elected an honorary member of the Turkish Society of Gynaecology.

The Spring Dinner of the Wessex Rahere Club will be held in the Royal Clarence Hotel, Exeter, on Saturday, April 18th. Further details may be had from Mr. A. Daunt Bateman, 11 The Circus, Bath.

EXAMINATION RESULTS

*UNIVERSITY OF BRISTOL*

*M.D.:* P. C. Farrant, K. R. Gough, M. D. Turner.

*Ph.D.:* H. S. M. Crabb, D. G. Lyon.

# Appointments

F. G. Bolton, M.D., M.R.C.P., Consultant Clinical Pathologist, Churchill Hospital, Oxford.
A. G. Freeman, M.A., M.D., M.R.C.P., Consultant Physician, Swindon and Cirencester area.
Joyce Fung, M.B., M.O. British Guiana.
Brenda Grant, M.B. (Liverpool), D.P.W., Supervising Psychiatrist, Gracie Square Hospital, New York.

*UNITED BRISTOL HOSPITALS*

*DECEMBER, 1958 TO FEBRUARY 1959*

| *Name* | *Appointment* | *Formerly* |
|---|---|---|
| EVANS, K. T., M.B., CH.B., M.R.C.P., D.M.R.D., F.F.R. | Consultant Radiologist. | Senior Registrar in Radiodiagnosis, Hammersmith Hospital & Postgraduate Medical School. |
| BARRITT, D. W., M.D., M.R.C.P. | Assistant Cardiological Physician. | Lecturer in Medicine, University of Bristol (Senior Registrar status). |

*SOUTH WESTERN REGIONAL HOSPITAL BOARD*

*DECEMBER, 1958 TO FEBRUARY, 1959*

*BRISTOL*

| *Name* | *Appointment* | *Formerly* |
|---|---|---|
| JOHNSON, D. H., M.D. (Bristol) | Consultant Pathologist, Weston-super-Mare General Hospital. | Lecturer in Pathology, University of Bristol. |
| POLEY, B. A., M.B., CH.B. (Bristol), D.A. | Anasthetice Registrar, Southmead Hospital. | S.H.O. Anaesthetist, United Bristol Hospitals. |
| WHITE, Miss C. M., MB., CH.B. (Glasgow), D.C.H. (London). | Paediatric Registrar, Southmead Hospital. | Paediatric Registrar, Nottingham Children's Hospital. |
| MITRA, A. K., M.B., B.S. (Calcutta). | Registrar to Dept. of Pathology Frenchay Hospital. | S.H.O. in Pathology, Bolton District General Hospital. |
| ROYLANCE, J., M.B., CH.B. (Bristol), D.M.R.D. | Registrar in Radiodiagnosis, Southmead Hospital. | Short service commission in R.A.M.C. Junior Specialist in Radiology. |

| *Name* | *Appointment* | *Formerly* |
|---|---|---|
| WEBB, A. J., M.B., CH.B. (Bristol). | Surgical Registrar, Cossham-Frenchay Group of Hospitals. | S.H.O. in General Surgery, Frenchay Hospital. |

*NORTH GLOUCESTERSHIRE*

| *Name* | *Appointment* | *Formerly* |
|---|---|---|
| CHIPPINDALE, F. W., N.B., B.S. (London), D.A. (England), F.F.A.R.C.S. | G.P. Anaesthetist, Dilke Memorial Hospital, Cinderford. | Royal Navy — Surgeon Commander. |
| WARD, F. W., M.B., B.S. (London). | Surgical Registrar, Cheltenham General Hospital. | Casualty Officer & Radiotherapy House Surgeon, Essex County Hospital. |
| CARDALE, J. D., M.B., CH.B. (Bristol). | G.P. Surgeon, Lydney & District Hospital. | In general practice in Lydney. |

*BATH*

| *Name* | *Appointment* | *Formerly* |
|---|---|---|
| KELLY, T. STUART-BLACK, M.B., CH.B. (Manchester), F.R.C.S.ED., D.O.M.S. | Consultant Ophthalmic Surgeon, Bath Eye Infirmary. | Consultant Ophthalmic Surgeon, Victoria Eye Hospital, Hereford. |
| WINTERTON, A. J., M.B., B.S. (Lond.), D.R.C.O.G., D.C.H. | G.P. Anaesthetist, Chippenham Hospital. | In general practice in Chippenham. |
| BEARD, Miss M. F., B.A., M.B., B.CHIR. (Cantab.) | Anaesthetic Registrar, Bath Group of Hospitals. | S.H.O. to Anaesthetic Dept., United Norwich Hospitals. |

*DEVON AND CORNWALL*

| *Name* | *Appointment* | *Formerly* |
|---|---|---|
| MONKS, P. J. W., M.B., B.S. (Lond.), F.R.C.S. (Eng.). | Consultant Surgeon, Devon and Exeter Clinical Area (Torquay). | Senior Surgical Registrar, United Bristol Hospitals. |
| PENCHEON, J. M., M.B., CH.B. (Leeds), D.P.M. | Consultant Psychiatrist, West Cornwall Clinical Area. | Assistant Psychiatrist, Lancaster Moor Hospital, Lancaster. |
| RODGER, W., M.A., M.B., B.CHIR. (Cantab.), D.P.M. | Consultant Psychiatrist, Devon & Exeter Clinical Area. | Senior Psychiatric Registrar, St. Bartholomew's Hospital and the North Middlesex Hospital, London. |
| MCCORMICK, A. J. A., M.B., CH.B. (Edin.), F.R.C.S. (Edin.), D.O.M.S. | Ophthalmologist, Torquay (School Eye Service). | Senior Ophthalmic Registrar, United Bristol Hospitals. |
| HALL, G. H., M.B., B.S. (London), M.R.C.P. (London). | Senior Medical Registrar, Royal Devon & Exter Hospital (joint appt. with the United Bristol Hospitals). | Medical Registrar, University College Hospital. |
| SIME, D. A., M.B., CH.B. (Edin.). | Senior Hospital Medical Officer Royal Western Counties Institution, Starcross. | Assistant Psychiatrist & Deputy Physician Supt. Aston Hall Mental Def. Hospital, Derbyshire. |
| HARRIS, R. H., M.B., B.S. (London), D.P.M. | Assistant Psychiatrist, Digby-Wonford Hospital, Exeter. | J.H.M.O. Moorhaven Hospital, Ivybridge, S. Devon. |
| ARTHUR, L. J. H., M.B., B.CHIR. (Cantab.), D.R.C.O.G., M.R.C.P. (London). | Clinical Asst. in Medicine, Tiverton District & Post Hill Hospitals, Tiverton. | In general practice in Tiverton. |
| JOHNS, E. R., M.B., B.S. (London), L.M.C.C., D.R.C.O.G. | Registrar in Obstetrics and Gynaecology, Redruth Hospital. | House Surgeon, Chase Farm Hospital, Enfield. |
| LEDWICH, J. R., M.B., CH.B. (Manchester). | Medical Registrar, Royal Cornwall Infirmary, Truro. | Resident Medical Officer, Oldham Royal Infirmary. |
| TANDON, P. N., M.B., B.S. (Lucknow). | E.N.T. Registrar, South Devon & East Cornwall Hospital, Plymouth. | S.H.O. in E.N.T. Surgery, Bristol General Hospital. |